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MECHANICAL CHARACTERISTICS OF THE HUMAN AIRWAY IN RELATION TO THE USE OF THE INTERRUPTER VALVE.

BY

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SUMMARY

1. Electrical and mechanical analogues have been used to formulate hypotheses concerning the mechanical characteristics of the respiratory tract, and these hypotheses have been confirmed by examination of "unscanned" pressure records obtained from the Clements' Valve during the repetitive interruption of airflow in man.

2. At normal respiratory frequencies, impedance is due mainly to the sum of resistance components. Impedance reaches a minimum at a frequency of about 30 c/sec, and small resonant peaks are seen at 6.6, 18.7, 28, and 56 c/sec.

3. Gas inertance and mouth wall compliance are important determinants of the rate of pressure equilibration between the lungs and the mouth.

4. Damping is normally less than critical. The secondary rise of pressure seen after flow interruption is due to continuing movement of the diaphragm compressing the lung gas volume against a tense chest wall.

5. If airway resistance is increased, damping may exceed the critical value, and 90% pressure equilibration may occupy 100 m.sec.

6. These findings are considered in relation to the use of the Clements' interrupter valve. The importance of a relaxed chest wall during the test is emphasized. Owing to the continuing action of the diaphragm, the pressure immediately following interruption normally gives the best estimate of airway resistance; however, where equilibration is slow, a late pressure is preferable.

The "scanning" zone of the Clement's interruptor valve represents one compromise between these two conflicting requirements; the possibility of speeding pressure equilibration by addition of an external inertance has yet to be exploited.

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INTRODUCTION

One of the earliest, and often the only clear symptom that results from mild or moderate respiratory exposure to an anticholinesterase vapour is the sensation of "tightness in the chest". This symptom is linked, at least in some degree, with "bronchospasm", and methods of measuring the increase of airway resistance following inhalation of an anticholinesterase thus offer one possible simple approach to the quantitation of respiratory exposure.

The method of airway interruption has been applied to the clinical measurement of bronchospasm for more than 30 years (1). It is a convenient technique for applied physiological work, since the apparatus can be quite portable (2), and little co-operation is required from the subject. A form of interrupter valve developed by Clements (3, 4 and Appendix I) and loaned to this Establishment for trial through standardization channels has been used to follow changes in airway resistance produced by inhalation of GB (3, 5) and CS (6). However, the accuracy of the results obtained has been questioned, and some have even stated that bronchospasm could be detected equally well by "the tossing of a coin" (7). Our data would not support such a categorical rejection of the method, but a considerable variation in the results was found both for a given subject, and from one normal subject to another.

Three assumptions are made when the interrupter valve is used to measure airway resistance:

(1) the pressure gradient from chest to valve, prior to interruption, is the simple resultant of components due to lung tissue, airway, and valve resistance.

(2) following interruption, mouth and chest pressures are rapidly equalized;

(3) chest pressures remain essentially unaltered during the course of equilibration.

The airway system has an impedance that is the resultant of many independent parameters, resistance, compliance, and inertance, arranged both in series and in parallel, and to study the validity of the above assumptions, it is convenient to represent this complex system by appropriate electrical and mechanical analogues.

In the present paper, findings from two such analogues are applied to the interpretation of Clements' interrupter valve tracings, both scanned (ref.4 and Appendix 1) and unscanned. The normal range of values is defined, and the effect of deliberate variations in chest and upper airway compliance, gas inertance, and upper airway resistance examined. When airway resistance is normal, the basic assumptions of the method are substantially valid, and the unscanned pressure immediately following interruption gives the best estimate of both airway resistance and also of an added external resistance. However, when airway resistance is increased, equilibration is slowed, and the final pressure reading is appreciably reduced by mouth compliance. In these circumstances, the "scanned" pressure has greater validity than the unscanned pressure, but neither method can give a fully quantitative measure of airway resistance.

THEORETICAL

Changes of intra-thoracic pressure during equilibration

Several factors may affect intrathoracic pressure following interruption of airflow at the mouth. It is convenient to examine these factors theoretically before considering use of the analogues, and of the valve in man.

(a) Continued respiratory movement. It is assumed that a steady movement of 0.5 l/sec is maintained by the diaphragm during the 50 m.sec. of flow interruption characteristic of the Clements valve; in essence, a closed system is compressed by 25 ml. Acting against the compliance of lung gas alone ($\sim$0.004 l/cm.H_2O), this would increase intra-thoracic pressure by more than 6 cm.H_2O; acting against the compliance of the relaxed chest wall ($\sim$0.200 l/cm.H_2O), the pressure rise would be only 0.13 cm.H_2O. In practice, continued respiratory movement produces a slow rise of pressure to a level between these extremes determined by the extent of chest relaxation.

(b) Gas inertance. During closure of the Clements' valve, flow falls from 0.5 l/sec to zero in about 12 m.sec. Assuming a gas inertance of 0.01 cm.H_2O/l/sec(8), the pressure within the airway would rise immediately by some 0.4 cm.H_2O. However, the volume of gas in which this pressure is developed (150 - 160 ml.) of dead space gas) is small, and although inertance contributes to the first oscillation of mouth pressure, it has a negligible influence on the final equilibrium pressure.

(c) Mouth compliance. During equilibration, the mouth compliance must "charge" to the same pressure as the alveolar gas volume. Mouth compliance is normally about a quarter of lung gas compliance (4), so that this factor reduces equilibrium pressure by some 20%; in absolute units, the effect of mouth compliance is much greater when airway resistance is increased.

The overall effect of these three factors is such that in subjects with a normal airway resistance, the equilibrium pressure is a little less than the initial gradient due to the sum of airway and lung tissue resistance. However, where airway resistance is increased, the equilibrium pressure tends to be considerably less than the initial pressure gradient, except in subjects with a tense chest.

METHODS

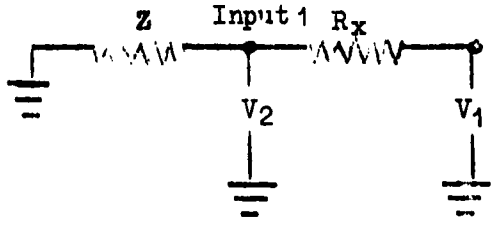
1. Electrical analogue of the respiratory system

The physiological components of the system, and their approximate electrical and mechanical equivalents are summarized in Table 1. In the electrical circuit (Fig.1), viscous resistances were represented by electrical resistances (scale 1 cm.H_2O/l/sec at a flow of 0.5 l/sec = 1 K ohm), compliances were represented by capacitances (scale 0.001 l/cm.H_2O = 1 μF), and inertances by inductances (scale 0.001 cm.H_2O/l/sec^2 = 1 H).

(a) Impedance to normal respiration. During normal respiration, the main forces are generated by the diaphragm; however, the chest muscles are usually sufficiently active to avoid paradoxical movement (that is, the compliance of the chest wall approaches zero during respiratory activity). This situation was represented in the electrical model by disconnecting tissue compliance C_c from tissue resistance R_c, and attaching a high output impedance sine-wave oscillator (Ediswan R.666) to input 1 ("external" to R_c). Respiration through the resistance of the open interrupter valve R_e was simulated by

connecting R_e to earth through the switch S_1.

The impedance Z of the analogue was determined by oscilloscope, voltages being measured across the analogue + small known resistance $R_x(V_1)$, and across the analogue alone (V_2):



High output inpedance Sine-wave oscillator

The ratio of peak to peak voltages $\frac{V_1 - V_2}{V_2}$ was proportional to the ratio R_x/Z, and the impedance of the analogue was thus given by $R_x.V_2/(V_1 - V_2)$. Resonant frequencies were determined by slowly varying the frequency of oscillation until "beats" were suppressed. Lissajou patterns were plotted by minimising R_x, and plotting $V_1 - V_2$ and V_2 on the two axes of the cathode ray screen.

(b) Response to interruption of flow. (i) Step change of flow. A "perfect" interrupter valve would give rise to an instantaneous interruption of a steady flow. It was not possible to simulate this situation using the entire electrical analogue, since the component representing lung compliance (C_L) would not transmit a steady flow. However, to a first approximation, lung pressures are not altered by flow interruption (9); thus for the purpose of studying equilibration rates and damping, a constant voltage d.c. potential could be applied to the lung gas compliance (Input 2 of Fig.1), and interruption of flow simulated by opening switch S_1. The "mouth" pressure (potential) during equilibration was observed on an oscilloscope, and also recorded on a high speed pen oscillograph (limiting frequency response 90 c/sec). (ii) Use of Clement's valve. The Clement's valve takes 12 m.sec. to complete the interruption of airflow. The electrical analogue was arrange as for (i), except that S_1 was kept closed, and the external resistance was increased from 6.8 to 100 K ohms over the space of 12 m.sec., using a potentiometer operated by a suitable cam.

2. Mechanical analogue of respiratory system

The mechanical analogue is shown in Fig.2. It differed from the electrical analogue in two main respects:

(i) non-linear resistances were used, and

(ii) continuing respiratory movement during interruption of flow was simulated by a steady input of air (0.5 l/sec) into the large tank (109 l. volume) representing lung gas, lung tissue, and chest wall compliance.

The airway was represented by a rigid tube of 50 cm length and 5 cm^2 cross-section, leading from the "lungs" to the interrupter valve. This provided adequate representation of the inertance and capacitance of gas in the airway; airway resistance was supplemented by an orifice R_a (2 - 8 mm. diameter orifices used in different experiments).

The mouth was shown as a small branch channel near the pressure tapping, leading through a 7 mm orifice R_m (mouth tissue resistance) to a rigid vessel of 1 l. capacity (mouth gas capacitance and tissue compliance).

"Mouth" pressures during equilibration were recorded from a piezometer ring, using a capacitance manometer and pen oscillograph (with alternative display on a cathode ray oscillograph).

In some experiments, the interrupter valve was set in the open position, and "step" interruptions of flow were obtained by rapid rotation of a tap. In other experiments, the Clements' valve was driven at the standard rate (interruption of flow over course of ~12 m.sec).

3. Human experiments

(a) Normal values. The airway resistance of 132 healthy servicemen, aged 17 - 42 yr, was measured by means of the Clements' interrupter valve, using the standard "scanning" ports, with independent measurement of flow and interruption pressures (Appendix I).

The standard procedure, followed by all subjects, consisted of a series of practice runs until the subject was able to maintain a flow of 0.5 l/sec (to within ± 10%) throughout expiration. Ten maximal expirations were then made at the standard rate, with intervals of 30 sec between successive tests. The airway resistance was calculated from the steady values of P_F and P_I during the middle third of each expiration.

In some subjects, additional tests of resistance were made during short expirations following a normal inspiration, and in others, the airway resistance was calculated for the middle third of a steady maximal inspiration.

(b) "Unscanned" interrupter valve records. The "scanning" ports of the Clements' valve were closed, and a continuous pressure record obtained from a piezometer ring (Appendix I).

Unscanned records were obtained during maximal expiration at 0.5 l/sec (10 observations on each of 13 subjects) and at 1.0 l/sec (10 observations on each of 10 subjects). Records were also obtained during maximal inspiration (1.0 l/sec, 10 subjects), and during breathing through an external resistance.

In 3 subjects experienced in respiratory manoeuvres, the pattern of the records was studied before and after deliberate modifications in the mechanical properties of the system. These included:

(i) decrease of chest wall compliance (deliberate tensing of chest muscles),

(ii) increase of airway resistance (deliberate constriction of glottis),

(iii) changes of airway gas inertance (breathing of mixtures containing 80% helium or 80% sulphur hexafluoride, and lengthening of dead space between mouth and valve),

(iv) increase of mouth compliance (1 l. bottle arranged as "shunt" in parallel with mouth compliance).

The effect of (iii) was also examined in two elderly subjects with chronic bronchitis.

(c) Estimation of external resistance. After the control value for airway resistance (R_A) had been determined from both scanned and unscanned records, an orifice with flow exponent similar to that for the respiratory tract (n = 1.6 - 1.7*) was interposed between the mouthpiece and the interrupter valve, and the total resistance (R_T) determined. The external resistance R_E was then calculated as the difference between these two values ($R_T - R_A$). Assuming that R_A is not altered by a small additional external resistance, the accuracy with which R_E is estimated provides a check on the success with which the method is measuring the resistance between lung and valve.

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*In the respiratory tract, the relationship between pressure P and flow dv/dt cannot be described by a single resistance constant R applicable to all flow rates. Resistance may be described by two constants R_α and R_β, thus:

$$P = R_\alpha \frac{dv}{dt} + R_\beta \left(\frac{dv}{dt}\right)^2 \text{, or}$$

over a limited and specified range of flows F, this may be simplified to a single resistance coefficient R and a flow exponent n, thus $P = RF^{(n)}$

In man, at flows of 0.5 - 2.5 l/sec, n = 1.6 - 1.7 (ref.3)

RESULTS

1. Using electrical analogue of respiratory system

(a) Impedance to respiratory flow. Under conditions of steady flow, the pressure gradient from chest to interrupter valve must be the simple resultant of resistance components due to lung tissue, airway, and valve resistance. The extent to which this conclusion must be modified under the phasic conditions of respiratory flow has been investigated by means of the electrical analogue and by complex analysis of the equivalent electrical circuit (Table 2 and Appendix II). At normal respiratory frequencies (0.25 - 1.0 c/sec), calculations predicted that the total sine-wave impedance ΣZ would be a little greater than the sum of the resistance components. There was also a moderate phase shift; detailed study of the complex analysis showed that this was attributable mainly to lung compliance at a frequency of 0.25 c/sec, and mainly to lung gas compliance at 1 c/sec. As the frequency was further increased, tissue inertance became progressively more important as a determinant of the overall properties of the system, and this component was responsible for the large positive phase angle at 100 c/sec.

Experimentally determined values for the impedance of the analogue (Fig.3) agreed with the results of complex analysis, within the limitations imposed by the electrical components. At both normal and high airway resistance settings, impedance declined from a figure close to the sum of resistance components at normal respiratory frequencies to a minimum at about 30 c/sec. With an airway resistance setting of 1.5K, resonance peaks were seen at 6.6 c/sec, 18.7 c/sec, 28 c/sec, and 56 c/sec; with the airway resistance setting increased to 15K, only slight displacement of these peaks occurred (resonant frequencies 6.5, 16.3, 30.2, 52, 99 c/sec). The impedance at resonant frequencies differed only slightly from that at non-resonant frequencies, suggesting that the system was almost critically damped. Lissajou patterns showed that under normal conditions "pressure" (voltage) and "flow" (current) applied at the "chest" (input 1) were in phase at a frequency of ~25 c/sec. The frequency for minimum area of the Lissajou pattern was not materially altered by large changes of R_a, C_m, and R_m, but was increased by a decrease of either lung gas compliance or lung tissue inertance:

	Frequency for minimum area of Lissajou pattern.
(a) normal settings	25.2 c/sec
(b) C_g = 2μF(normally 4μF)	36.0 c/sec
(c) L_1 = 5H (normally 10H)	34.0 c/sec

(b) Response to interruption of flow. (i) Step change of flow. With a constant d.c. potential coupled to lung gas compliance C_g (input 2 of Fig.1), the sudden interruption of flow by opening of switch S_1 reduced the analogue to a simple series RCL circuit.

The damping ratio d (see appendix III) for such a circuit may be calculated quite simply ($d = \frac{R}{2}\sqrt{\frac{C}{L}}$), and a 90% equilibration time may also be derived on theoretical grounds (Table 3); the latter applies to recovery from a simple constraint, and does not take account of the fact that "gas" is already flowing through the main inertance of the circuit prior to interruption. The 90% equilibration times are thus in practice shorter than the theoretical prediction.

The response of the analogue is illustrated in Fig.4. Damping was normally equal to or slightly in excess of critical, with a 90% response time of about 10 m.sec (Fig.4a). If mouth compliance or mouth tissue resistance was reduced (Fig.4b), the system became underdamped, and interruption of flow gave rise to an immediate chain of oscillations (90% response time effectively zero). On the other hand, equilibration was progressively slowed by increase of airway resistance (Fig.4c) or mouth compliance (Fig.4d). (ii) Use of interrupter valve cam. The results were essentially similar when flow was interrupted by the cam shaped to simulate the interrupter valve. The system was normally close to critical damping. With a low mouth resistance, the system became underdamped, and with a normal mouth resistance and increased airway resistance (15K), equilibration was not completed within the period of interruption. Both with normal and increased airway resistance, damping was increased by an increase of mouth compliance.

2. Using mechanical analogue of respiratory system

Experiments relating to the rate of equilibration following interruption of flow were repeated on the mechanical analogue, to examine the effect of replacing linear by non-linear resistors.

(i) "Step" interruption of flow. The observed damping ratios (calculated from the second and third half waves of the oscillation) or 90% equilibration times (where damping >critical) are shown in Table 4. Interpolating from this table, it may be concluded that with a combined mouth and airway resistance of 6.5 cm H_2O/l/sec as in normal man (airway orifice 6 - 7 mm diameter), the damping of the mechanical analogue was no more than 0.25 critical. At a similar setting, it was calculated that the electrical analogue was critically damped.

(ii) Use of interrupter valve. Results were similar to those obtained with a "Step" interruption of flow. At normal settings, damping was ~0.25 critical, and the transition to damping in excess of critical did not occur until the diameter of the airway orifice R_a was reduced to 4 - 5 mm.

3. Human experiments

(a) Normal values for Clements interrupter valve

(i) Interpretation of records. Pressures developed at the interruption pressure and flow pressure tappings of the Clements valve are commonly recorded by low frequency gauges. Using a high frequency recording system, oscillations can be seen in both interruption and flow pressure tracings (Fig.5). These are of different etiology to the oscillations discussed above, and reflect leakage between the pressure tappings and the body of the valve.* Since the flow pressure tapping was operating below mean valve pressure, and the interruption pressure tapping was operating above mean valve pressure, the minima of the "flow" tracing, and the maxima of the interruption pressure tracing were used in the calculation of pulmonary resistance. If low frequency gauges had been used, the apparent interruption pressure would have been 4 - 5 mm H_2O lower, and the apparent flow rate 2 - 3 l/min higher.

(ii) Distribution of normal values. Airway resistance, measured during a steady maximal expiration at approximately 0.5 l/sec, showed a slightly skewed distribution (Fig.6). The mean value was 2.60 cm H_2O/l/sec, with a standard deviation of 0.98 cm H_2O/l/sec, and 12/132 apparently healthy subjects yielded values greater than 4 cm H_2O/l/sec. The mean standard deviation of 10 successive measurements on the same subject was 0.81 ± 0.40 cm H_2O/l/sec (coefficient of variation 33.1 ± 16.9%).

*Leakage of a similar order was found in two samples of the valve, even after careful greasing.

(iii) Repetition of the test. Observations repeated at ½ min intervals remained constant (Table 5). There was no build-up of airway resistance with repetition of the test.

(iv) Length of expiration. Comparison of measurements made during a short expiration following a normal inspiration with the standard maximal expiration technique showed that the "short-puff" resulted in a lower value for airway resistance than the standard technique ($\Delta \pm$ S.E. in 10 subjects 0.50 ± 0.20 on $H_2O/l/sec^2$). The coefficient of variation was marginally greater than with maximal expiration (C.V. for 10 subjects 31.1%, compared with 25.4% for the standard technique on this group of subjects).

(v) Inspiratory tests. In 9 normal subjects the airway resistance during a standard maximal expiration was compared with the resistance measured during a steady maximal inspiration at the same rate (Table 6). In 3 subjects the resistance was significantly greater during inspiration than during expiration, in 2 it was significantly less, and in the remaining 4 there was no difference.

(b) Unscanned records from Clements' interruptor valve

(i) Form of tracing. The unscanned type of record is necessary to study the rate of pressure equilibration at the mouth following rapid repetitive interruption of airflow. The pattern of tracing observed usually conformed with that described by earlier authors (10) - a sudden rise of pressure at interruption, an underdamped oscillation, and a secondary phase of progressively increasing pressure (Fig.7). Comparing inspiratory and expiratory records, damping was much greater during expiration than during inspiration; furthermore, as the last 500 - 1000 ml of gas were expelled from the chest (final 1 - 2 sec of expiration), the secondary phase of increasing pressure often became very pronounced. In the example of Fig.7 (a normal subject), the pressure 50 m.sec after interruption was 35 - 40 mm H_2O, compared with 15 - 20 mm H_2O in the first few m.sec after interruption of flow. Damping ratios* for a group of 9 normal subjects are given in Table 6; in general, the records were underdamped during both inspiration and expiration and the observed values lay between ratios predicted from the electrical analogue (linear resistors) and the mechanical analogue (non-linear resistors).

*Here calculated from the logarithmic decrement ratio, see Appendix III.

(ii) Modification of tracing. The form of the tracing, and in particular the degree of damping could be modified by many of the procedures producing similar effects in the electrical and mechanical analogues. In normal subjects, the degree of damping could be increased and equilibration of pressures slowed by voluntary narrowing of the glottis, and an even more marked picture of delayed equilibration was seen in the two subjects where airway resistance was increased by chronic bronchitis (Fig.8d, cf. normal Fig.8a). The interposition of added gas inertance between the mouth and the valve (a 50 cm length of 5 cm^2 cross-section rigid walled tube) markedly decreased damping; in normal subjects, violent oscillations were seen (Fig. 8b), and damping was less than critical even when the glottis was deliberately narrowed to increase airway resistance (Fig.8c). Even in the patients with greatly increased airway resistance, pressure equilibration became possible during the period of airflow interruption (Fig.8e). A similar decrease of damping could be produced without increasing the external dead space if a few breaths of a very dense gas mixture (80% SF_6, 20% oxygen) were breathed immediately before the test; conversely, a few breaths of a low density mixture (80% helium, 20% oxygen) caused damping in excess of critical even in normal subjects. Damping was also increased by adding an external shunt compliance (glass vessel of 1 l. capacity); the effect was greatest when this was linked to the external dead space via a narrow orifice.

The importance of a relaxed chest wall to the constancy of chest pressure after interruption has already been noted theoretically. This point was tested experimentally in three subjects experienced in respiratory manoeuvres. Expiration was commenced with the chest wall held tense, and at a given signal the chest muscles were relaxed, expiration being maintained by the abdominal muscles alone. When the chest muscles were relaxed, the record showed little secondary rise of pressure (Fig. 9), but when the chest wall was tensed, pressures rose by 20 - 30 mm H_2O during the interrupted phase of the valve cycle, and the pressure over the "scanning" period (17 - 33 m.sec after interruption of flow) was correspondingly too high.

From the foregoing, a slow rise of pressure after interruption of flow may be due to either delay in equilibration or continued respiratory movement against a tense chest wall; however, the first is reduced or abolished by an added external inertance, whereas the second is unaffected by this manoeuvre.

(iii) Magnitude of "unscanned" resistance. The method of calculating airway resistance from the unscanned pressure records was that of Otis et al.(10), the interruption pressure being found by extrapolation of the "plateau" following the oscillations back to the instant of flow interruption. This method is valid in normal subjects, where the tracing is underdamped, but would give an erroneous answer in subjects where equilibration was slowed (as in Fig.8d). Comparison with the resistance calculated from standard "scanned" records in 13 subjects showed a significant discrepancy, the mean "unscanned" value for this group being 2.41, S.D. $\pm$ 1.04 cm $H_2O/l/sec$, and the mean "scanned" value 3.36, S.D. $\pm$ 1.30 cm $H_2O/l/sec$ ($\Delta \pm$ S.E. 0.95 $\pm$ 0.27 cm $H_2O/l/sec$, $0.01 > P > 0.001$). In 6 of the 13 subjects, the "scanned" measurements indicated a rather high airway resistance (>3.5 cm $H_2O/l/sec$); in 5 of these 6, the apparent high resistance was an artefact due to a secondary rise of pressure following interruption of flow, and the high resistance was not confirmed by "unscanned" measurements.

The average coefficient of variation of 10 consecutive "unscanned" readings on the same subject was 18.9 $\pm$ 5.2%. The corresponding coefficient for "scanned" readings on the same group of subjects was 22.2 $\pm$ 11.6%.

(iv) Effect of lung volume. Various laboratories making scanned measurements with the Clements valve have reported a large effect of lung volume on the calculated airway resistance (2, 11), particularly during the last 500 - 1000 ml of expiration. To examine how far this effect was due simply to tensing of the chest muscles at the end of expiration, the analysis was repeated using "unscanned" records. An effect of lung volume was still observed, but resistance rose much less steeply than with the "scanned" measurements during the terminal part of expiration (Fig.10).

(c) Estimation of external resistance

(i) During expiration. In the first series of 13 subjects, "scanned" measurements of total resistance were made before and after introduction of resistances of 2, 8, and 28 cm H_2O/l/sec, with a steady expiratory flow of 0.5 l/sec. The respective estimates of external resistance from these figures were 0.8, 4.6 and 10.2 cm H_2O/l/sec.

In a second series of 13 subjects, both scanned and unscanned measurements were made at a steady expiratory flow of 0.5 l/sec, using an external resistance of 3.9 cm H_2O/l/sec; the respective estimates of this resistance were 2.35, S.E. $\pm$ 0.66 and 2.82, S.E. $\pm$ 0.41 cm H_2O/l/sec by the two techniques.

A final series of 10 subjects were tested at a more rapid flow rate (1 l/sec); using an orifice with a resistance of 7.1 cm H_2O/l/sec at this flow rate, the respective estimates were 5.30, S.E. $\pm$ 0.50 cm H_2O/l/sec (scanned) and 7.55, S.E. $\pm$ 0.26 cm H_2O/l/sec (unscanned measurements).

(ii) During inspiration. In 10 subjects, unscanned measurements were made at steady inspiratory flow rates of 0.5 l/sec and 1.0 l/sec, using orifices with resistances of 4.6 cm H_2O/l/sec and 7.1 cm H_2O/l/sec at the two flow rates. Both resistances were estimated correctly, within the limits of experimental error (4.81, S.E. $\pm$ 0.23, and 7.64, S.E. $\pm$ 0.37 cm H_2O/l/sec).

DISCUSSION

1. Information from analogues of the respiratory tract

(a) Previous use of analogues. Several previous authors (4, 12, 13) have used electrical and/or mechanical analogues in an attempt to define the mechanical properties of the respiratory tract. However, previous models and analogues have omitted one or more important components - the mouth (9, 12, 13) gas inertance (9, 12), or the inertance of the abdominal and thoracic viscera (8), and the method of activation - either a sine-wave oscillator (12, 13) or a square wave (4, 9) - seem an incomplete representation of the events involved in use of the interrupter valve.

(b) Limitations of present analogues

(i) Knowledge of components. The normal values for many components of the present analogue - R_e, R_a, C_g, and C_l - are well established, and require no specific comment. C_m and R_m were determined by Clements, using a modification of the interrupter valve system (4). The present author has found static mouth compliance to vary quite widely from 0.4 to 4.8 ml/cm H_2O in normal subjects

(unpublished data). Chest wall compliance C_c has been obtained as the difference between the total compliance of the lungs and chest of 0.1 l/cm H_2O (4), and lung compliance C_l - that is, about 0.2 l/cm H_2O. As discussed in the theoretical section, if the chest wall is not relaxed following interruption, C_c many fall to 1/20th of this value. R_l has been estimated as 0,2 cm H_2O/l/sec at 0.5 l/sec flow from the difference between body plethysmograph and oesophageal measurements of resistance (15). $R_l + R_c$ is known from the difference between total thoracic and airway resistance (16) to be at least 1 cm H_2O/l/sec, and possibly 2 cm H_2O/l/sec. The inertance terms L_a and L_l have been investigated by measuring accelerative pressures at differing ambient pressures (8); the observed value of 0.01 cm H_2O/l/sec^2 at normal ambient pressures seemed due largely to the L_a component, and conformed with a theoretical prediction of L_a based on the dimensions of the respiratory tract.* Other estimates of overall inertance have been based (12) on

(i) comparisons of reactance at differing frequencies (0.04 cm H_2O/l/sec^2), and (ii) determination of resonant frequency (0.006 cm H_2O/l/sec^2); however, these calculations contain a number of uncertainties/.

The present analogues have been designed to operate over the range imposed by these uncertainties. Errors in the choice of values for L_l and R_c are of minor importance in determining the overall impedance to pressures applied at the mouth, or the rate of equilibration of lung and mouth pressures, but L_l plays an important part in controlling the response to high frequencies applied at the chest.

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* The theoretical prediction is based on a plane wave front. According to Mead (8), inertance would be twice the predicted value of 0.012 cm H_2O/l/sec^2 under conditions of laminar flow, and 1.22 times the predicted value with turbulent flow. Van den Berg (13) points out that the factors should properly be 1.00 for laminar flow and 1.33 for turbulent flow.

/ Dubois et al (12) state that the phase shift at 2.9 c/sec is -33°, and at 5.8 c/sec is zero (resonant frequency). These figures apparently refer to the phase angle between pressure and flow across the chest. Allowance is made for "the compressibility of alveolar air" in the case of pressures applied through a Drinker respirator, and for the "compressibility of air in the flowmeter and tracheobronchial tree" when pressure is applied at the mouth. However, no allowance is made for the compliance and resistance of the mouth, and none for the inertance of gas in the airway. The inertance calculated is thus somewhat uncertain.

(ii) Other limitations. The analogue is a single channel device, whereas the lung contains a large number of parallel bronchial pathways. However, it is not necessarily an advantage to multiply the number of channels, as does Van den Berg (13), without more knowledge of the relative distribution of resistance, capacitance, and inductance between channels.

The assumption is made that the walls of the air passages are rigid; in fact, the resistance increases throughout expiration, but the change of resistance over the middle range of lung volumes is not sufficient to invalidate results based on average resistances (2, 11).

Finally, in the electrical (but not in the mechanical) analogue, resistance is considered a linear function of flow. The damping ratio observed in human subjects is less than in the electrical model, and more than in the mechanical model; this could be due to part to acceptance of a rather low value for C_m, but is probably due in the main to a combination of a linear mouth resistance R_m and a non-linear airway resistance R_a.

(c) The natural resonant frequency.

The definition of resonance has caused some confusion in the past. Some authors (12, 17, 18) have considered the resonant frequency as that frequency at which pressure and flow were in phase. In the experiments of DuBois et al (12), with pressures applied at the mouth, resonance thus defined occurred at a frequency of 5 - 7 c/sec. In dogs (16), with pressures applied to the chest wall via a Drinker respirator, a similar "resonant" frequency was observed. In cats (13), with pressures applied to the trachea, the "resonant" frequency was somewhat higher (7 - 12 c/sec). In the present electrical model, with potentials applied to the chest, pressure and flow were in phase at a much higher frequency (25 c/sec) than in the dog, suggesting that L_1 may have been greater in life than in the model. Van den Berg (13) considered the resonant frequency as that frequency where impedance was at a minimum, and applying the input to the "trachea" of his model he described resonance at 6.6 and 18.7 c/sec; in the present model, with the input applied to the chest wall only, one well defined minimum of impedance occurred, at about 30 c/sec. Neither of the above definitions of resonance are as sensitive as the suppression of beats in a damped system; using this last technique, the present author has found resonance at 6.6, 18.7, 28, and 56 c/sec with normal settings of the analogue.

The primary frequency of interruption (10 c/sec) is well removed from a zone of resonance, and resonance in phase with interruptions is thus unlikely to affect the validity of the method. The only oscillations seen in the human experiments have a very high frequency (80 - 90 c/sec). Similar oscillations are seen when flow through the mechanical and electrical models is interrupted by the "Clements valve" (Figs. 5 and 6), although in the electrical model the frequency of oscillation (50 - 60 c/sec) conforms to the upper resonance band previously defined. Presumably these oscillations occur locally between the mouth and lung gas compliance, but in the mechanical model (and in man) the frequency of oscillation is increased by some contribution from chest-wall compliance.

(d) The rate of pressure equilibration

Many previous workers have considered the lungs and airway as a simple resistance/capacitance system, and have obtained "time constants" by multiplication of airway resistance and lung gas compliance; from such calculations it has been concluded "that 18 m.sec was a conservative estimate of 50% equilibration time" (18), and that 20 msec was required for equilibration (19). Others (2, 20) considered equilibration to be complete in 5 m.sec. The present work suggests that in the use of the interrupter valve, the compliance of the mouth has greater relevance than the compliance of lung gas. Further, the system is not a simple resistance/capacitance network, and gas inertance cannot be neglected in calculating rates of pressure equilibration. Indeed, the system is normally underdamped; oscillations therefore occur, and equilibration is essentially instantaneous. When airway resistance is increased, damping exceeds critical and equilibration times become lengthy (9, 19, 21). In these circumstances, the equilibration pressure may not even be indicated by the scanning phase of the Clements valve (17 - 33 m.sec after interruption); there has recently been a plea for measurements as late as 80 m.sec after interruption (19), but at this time it is difficult to distinguish slow equilibration from secondary pressure changes, and the alternative of speeding equilibration by use of an external inertance seems preferable.

(e) Addition of external resistance

The addition of an external resistance is not fully comparable with an increase of airway resistance, since the constriction is distal to the "shunt" represented by the mouth. In particular, the external resistance does not alter the damping of the lung/mouth circuit in the way that an increase of airway resistance would. However, under steady flow conditions prior to interruption, the pressure gradient is similar whether the resistance is internal or external. Further, the 20% reduction of pressures by mouth compliance (see theory) does not affect the pressure drop across the external resistance. Thus the equilibrium pressure after flow interruption should be proportional to the sum of airway and external resistances, unless the external resistance has modified the airway resistance (as by altering the tendency of the airway to collapse during expiration).

2. Normal values for airway resistance in relation to technique

(a) Absolute magnitude.

The absolute magnitude of airway resistance, as determined by interrupter valve, is greater than values obtained by oesophageal balloon (6, 19, 22), and much greater than by body plethysmograph (22).

The results of the present interrupter valve series are higher than some previously reported (6). This may be due in part to the use of a long, steady expiration, as smaller, but more variable values were obtained after a "short puff" through the interrupter valve. Full expiration could have led to progressive collapse of the airway, but repetitive measurements of resistance did not show any such trend (Table 5). The effort of controlling flow at a steady rate probably led to tensing of the chest wall in some subjects, with consequent over-estimation of resistance when "scanned" pressure records were used. Finally, it is likely that alveolar units with a low resistance to airflow had emptied before the midpoint of expiration was reached. The "average" resistance seen at mid-expiration would thus be weighted in favour of higher resistance units. If it is intended to use the valve as a precise and fully quantitative measure of airway resistance, this last point is a valid criticism of the maximal expiration technique. However, differences of resistance between units are more likely to occur in the presence of bronchospasm, and the method of maximal expiration thus helps to bring out the difference between normal and abnormal subjects.

(b) Reproducibility of resistance readings

It has been stated (5, 23, 24) that changes of airway resistance are difficult to demonstrate by means of the interrupter valve because of variation in control measurements, and a coefficient of variation as great as 50% has been quoted (5). This last assessment was based on portable apparatus fitted with water gauges. Leakage at the valve was thus difficult to assess, lung volume was not known, and it was necessary to calculate airway resistance from the difference between a pair of oscillating water menisci. The present figure of 18.9% coefficient of variation for unscanned readings compares favourably with the body plethysmograph (C.V. 24.7%, ref. 22), and the oesophageal balloon, (C.V. 27.2%, ref. 6).

(c) Use of resistance coefficient.

The calculation of "resistance" for a non-linear component is of doubtful propriety, and some physicists have preferred to calculate the "constant" R in the equation $P = RF^{(n)}$ (page 6) assuming a constant exponent n(2). However, the resistant term R is still not a true constant, for the exponent n varies with the structure of gas flow in the respiratory tract. During resting ventilation (3) , turbulence may develop in the trachea and upper airway, but it is not present in the main resistance elements of the airway, except at points of branching. The apparent constant n is thus a composite figure embracing laminar flow through some resistance elements (n = 1), and turbulent flow (n = say 1.85) through the remaining resistance elements. The values of n to be expected at different flow rates are shown in Table 8. In practice, n is somewhat higher (1.5 - 1.7) over the range of the interrupter valve measurements (0.5 - 2.5 l/sec). owing to the effects of branching; however, at low flow rates n inevitably tends towards unity, and at high flow rates to a limiting value of 1.85. Thus, however resistance is expressed, it must be confined to a specified flow range, and having accepted this premise, the "resistance coefficient" offers no advantage over the simpler conventional unit of physiology (cm H_2O/l/sec at 0.5 l/sec flow).

(d) Measurement of external resistance.

The procedure of estimating an external resistance has previously been used to validate the interrupter valve technique (2). On the basis of this procedure, it can be concluded that (i) each of the several procedures adopted gives an index of increased resistance, (ii) a 1 l/sec expiration is preferable

to an 0.5 l/sec expiration, (iii) inspiration is preferable to expiration, and (iv) early unscanned readings are preferable to scanned readings in the estimation of an added external resistance.

The explanation of these findings is probably that the external resistance modifies the secondary pressure rise; either expiration is slightly slower, or subjects find it easier to maintain a steady flow without tending the chest when an external resistance is present than when it is not. Conclusion (iv) is not applicable to an increase of resistance internal to the mouth, since equilibration is then slow. When resistance is increased, a scanned reading (or a late unscanned reading, 19) is more likely to give the correct resistance value than an early unscanned reading.

CONCLUSION

Whether the scanning device is used or not, with the Clements interrupter valve a fixed interval between interruption of flow and measurement of pressure is desirable on grounds of objectivity in interpreting tracings. An early unscanned reading will be correct under normal conditions, but very unsatisfactory in the presence of bronchospasm. The scanning position of the Clements valve (17 - 33 m.sec), although determined primarily by engineering considerations, probably represents a useful compromise for the estimation of both normal and increased resistance. The possibility of speeding equilibration by addition of an external inertance in subjects with bronchospasm has yet to be exploited.

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TABLE 1

Components used in Electrical and Mechanical Analogues of the Resp

Abbreviation	Component	Normal units, at flow of 0.5 l/sec*	Electrical Analogue	Electric (sine-wa	
				10 c/sec	1
R_e	External (flow measuring) resistance.	6.8 cm H_2O/l/sec	6.8K Ω	6.8K	
R_m	Mouth tissue resistance.	5.0 cm H_2O/l/sec	5.0K Ω	5.0K	
R_a	Airway resistance.	1.5 cm H_2O/l/sec	1.5K Ω	1.5K	
R_l	Lung tissue resistance.	0.2 - 0.5 cm H_2O/l/sec	0.5K Ω	0.5K	
R_c	Chest wall and extra thoracic resistance.	0.5 - 1.5 cm H_2O/l/sec	0.5K Ω	0.5K	
C_m	Compliance of mouth tissues.	0.001 l/cm H_2O	1μF	15.9K	
C_l	Compliance of lung tissues	0.200 l/cm H_2O	200μF	0.08K	
C_c	Compliance of chest wall and extra-thoracic tissues	0.200 l/cm H_2O	200μF	0.08K	
C_g	Compliance of alveolar gas.	0.004 l/cm H_2O	4μF	4.0K	
L_a	Pulmonary gas inertance	0.01 cm H_2O/l/sec^2	10 H	0.6K	
L_c	Inertance of lungs, chest and extra thoracic tissues	0.01 cm H_2O/l/sec^2	10 H	0.6K	

*It is necessary to specify the flow rate on account of the non-linearity c

1

TABLE 1

s used in Electrical and Mechanical Analogues of the Respiratory System

	Normal units, at flow of 0.5 l/sec*	Electrical Analogue	Electrical impedance (sine-wave potential)			Mechanical analogue
			10 c/sec	100 c/sec	1 c/sec	
suring)	6.8 cm H_2O/l/sec	6.8K Ω	6.8K	6.8K	6.8K	Clements valve(open)
stance.	5.0 cm H_2O/l/sec	5.0K Ω	5.0K	5.0K	5.0K	7mm orifice(= 2.8 cm H_2O/l/sec)
	1.5 cm H_2O/l/sec	1.5K Ω	1.5K	1.5K	1.5K	8mm orifice(= 2 cm H_2O/l/sec)
tance.	0.2 - 0.5 cm H_2O/l/sec	0.5K Ω	0.5K	0.5K	0.5K	Not represented.
tra ce.	0.5 - 1.5 cm H_2O/l/sec	0.5K Ω	0.5K	0.5K	0.5K	Not represented.
th	0.001 l/cm H_2O	1μF	15.9K	1.59K	159K	1 l vessel
g tissues	0.200 l/cm H_2O	200μF	0.08K	0.008K	0.8K	109 l vessel
st wall tissues	0.200 l/cm H_2O	200μF	0.08K	0.008K	0.8K	
eolar	0.004 l/cm H_2O	4μF	4.0K	0.4K	40.0K	Not represented
rtance	0.01 cm H_2O/l/sec^2	1C H	0.6K	6.0K	0.06K	Tube 50 cm x 5 cm^2
s, chest tissues	?0.01 cm H_2O/l/sec^2	10 H	?0.6K	?6.0K	?0.06K	Inflow to 109 l reservoir of 0.5 l/sec during interruption.

o specify the flow rate on account of the non-linearity of airway resistance.

2

TABLE 2

Calculated values of resistance ΣR (including external resistance R_e of 6.8K), reactance $\Sigma (X_L - X_c)$, impedance ΣZ, and phase angle ϕ for analogue circuit, with airway resistance of (a) 1.5K, and (b) 15K.

f	Airway resistance normal (1.5K)				Airway resistance high (15K)			
	ΣR	$\Sigma(X_L-X_c)$	ΣZ	ϕ	ΣR	$\Sigma(X_L-X_c)$	ΣZ	ϕ
0.25 c/sec	9.2 K	-3.67K	9.9 K	$-21^{\circ}48'$	22.2 K	- 6.29K	23.1 K	$-15^{\circ}51'$
1	8.81K	-2.57K	9.18K	$-16^{\circ}17'$	17.7 K	-10.1 K	20.8 K	$-29^{\circ}41'$
10	2.46K	-2.37K	3.42K	$-43^{\circ}54'$	1.73K	- 3.29K	3.74K	$-62^{\circ}14'$
100	1.02K	+5.57K	5.67K	$+79^{\circ}37'$	1.00K	+ 5.59K	5.68K	$+79^{\circ}52$

TABLE 3

Calculated damping ratio and 90% equilibration time of constant lung potential, following opening of switch S_4 in analogue. Fixed mouth tissue resistance of 5K, and fixed lung gas inertance of 10H assumed, selected settings of airway resistance and mouth compliance as shown.

Airway resistance	Damping ratio			90% equilibration time		
	Mouth compliance			Mouth compliance		
	0.5μF	1.0μF	2.0μF	0.5μF	1.0μF	2.0μF
1.5K	0.73	1.05	1.46	Oscillations	13 m.sec	
3.9K	1.00	1.41	1.99	9 m.sec		
10.0K	1.68	2.37	3.35			
15.0K	2.24	3.16	4.47			
25.0K	3.35	4.75	6.71			
39.7K	5.0	7.07	10.0			107 m.sec
58.3K	7.07	10.0	14.0		76 m.sec	
84.4K	10.0	14.0	20.0	54 m.sec		

TABLE 4

Damping ratio and 90% equilibration time of mouth pressure in mechanical analogue of airway at selected values of airway resistance. Mouth resistance 2.8 cm H_2O/l/sec at 0.5 l/sec, mouth compliance 0.001 l/cm H_2O, airway gas inertance 0.012 cm H_2O/l/sec^2 for plane wave front.

Orifice Diameter	Ra(cm H_2O/l/sec at 0.5 l/sec)	Damping ratio	90% equilibration time
8 mm	2.0	0.12 critical	oscillations
7	2.8	0.15 critical	oscillations
6	8.0	0.27 critical	oscillations
5	12.8	0.33 critical	oscillations
4	28.0	> critical	53 m.sec
2	320.0	> critical	350 m.sec

TABLE 5

Ten successive measurements of airway resistance at mid-point of steady maximal expiration. Interval between measurements 30 sec. Pooled results for first 33 subjects of present series.

Measurement	Airway resistance (cm H_2O/l/sec)	S.E. of mean
1	2.74	± 0.19
2	2.66	± 0.18
3	2.98	± 0.25
4	2.68	± 0.17
5	2.50	± 0.26
6	2.63	± 0.18
7	2.84	± 0.26
8	2.81	± 0.18
9	2.90	± 0.23
10	3.04	± 0.22

TABLE 6

Comparison of expiratory and inspiratory resistance in 9 normal subjects. Mean and S.E. of difference shown.

Expiratory resistance (cm H_2O/l/sec)	Inspiratory resistance (cm H_2O/l/sec)	$\Delta \pm$ S.E.
3.48	2.66	-0.82 ± 0.33
1.26	4.45	$+3.19 \pm 0.30$
3.09	3.14	$+0.05 \pm 0.30$
3.74	2.69	-1.05 ± 0.67
1.22	3.67	$+2.45 \pm 0.19$
2.47	3.10	$+0.63 \pm 0.32$
1.55	2.54	$+0.99 \pm 0.30$
1.42	1.31	-0.11 ± 0.18
3.58	2.43	-1.15 ± 0.51

TABLE 7

Observed damping ratios in normal subjects

Inspiration	Expiration
0.41	0.70
0.24	0.21
0.18	0.36
0.30	0.30
0.07	>1.00
0.27	0.70
0.24	0.70
0.16	>1.00
0.17	0.40
Mean $\pm$ S.E. 0.23 ± 0.03	0.60 ± 0.03

TABLE 8

Percentage of "laminar" and "turbulent" resistance in the human airway at selected flow rates, assuming Reynold's number of 2000, and average flow exponent for system as a whole.

Flow l/sec	"Laminar" resistance ($R = KF$)	"Turbulent" resistance ($R = KF^{1.85}$)	Average exponent n for entire airway ($R = KF^{n}$)
0.4	100 %	0 %	1.00
0.5	81.7	18.3	1.20
0.6	44.1	55.9	1.53
0.8	42.5	57.5	1.55
1.4	8.3	91.7	1.80
5.3	0.4	99.6	1.84
10.0	0	100.0	1.85

APPENDIX I to P.T.P. 829
The Clements Interrupter Valve

The Clements valve (4) is a development of an earlier system for measuring airway resistance during single interruptions of airflow (3), Airflow is interrupted 600 times per minute, flow being deduced from the pressure prior to interruption and chest pressure from the pressure during interruption. Normally, pressure within the valve is "scanned" by two ports rotating with the valve, and only the middle thirds of "flow" and "interruption" pressures are transmitted to the recording system; this is intended to permit the use of low frequency recording systems.

The apparatus consists basically of two carefully machined concentric metal sleeves and an outer hood (Fig.11). The outer sleeve carries four longitudinal slits, and there are four corresponding but narrower slits in the inner sleeve. The inner sleeve is rotated at a constant rate (150 r.p.m.). As the sleeve turns, interruption of flow is developed over the course of 12 m.sec; interruption is then maintained for some 50 m.sec, and flow is restored over the following 12 m.sec. The "scanning" ports are arranged to communicate with the interior of the valve 17 - 33 m.sec after interruption of flow, and 17 - 33 m.sec after restoration of flow. The purpose of the outer hood is to shape the airstream during the flow phase of the valve cycle so that the pressure developed during this phase is proportional to the 1.66th power of flow rate (thus matching the overall characteristics of the respiratory tract).

In some devices incorporating the Clements valve flow pressures (P_F) and interruption pressures (P_I) are subtracted mechanically from each other; however, in all the present experiments where the "scanning" ports were used the two pressures were recorded independently by capacitance manometers and high frequency pen oscillographs. The "scanning" device necessarily obscures details of the pressure during equilibration, and thus for the purpose of comparison with results from the electrical and mechanical analogues, the normal "scanning" ports were closed, and instead a continuous ("unscanned") pressure record was obtained from a piezometer ring mounted proximal to the interrupter valve proper.

APPENDIX II to P.T.P.829

Calculation of Impedance of Lung-Airway analogue by complex analysis

Complex analysis is a mathematical procedure whereby a complex series/parallel resistance/capacitance/inductance network is reduced to an equivalent series resistance and capacitance or inductance at a given frequency.

Consider first the series case. The impedance X_S to a pure sine wave voltage of frequency f is given by

$$Z_S = \sqrt{\Sigma R^2 + \Sigma (X_L - X_C)^2} \quad \ldots\ldots\ldots (1)$$

where ΣR is the total resistance of the series segment of the circuit, X_L the inductive reactance ($X_L = 2\pi fL$, where L is the inductance), and X_C the capacitative reactance ($X_C = 1/2\pi fC$) where C is the capacitance). Addition of individual series elements is simplified by use of the j operator to signify the 90^o difference of phase between resistance and reactance ($j = \sqrt{-1}$):

$$Z = R + j (X_L - X_C) \quad \ldots\ldots\ldots (2)$$

If written in this form, addition of series elements may be carried out directly:

$$\Sigma Z_S = Z_{S1} + Z_{S2} \quad \ldots\ldots\ldots (3)$$

In the case of circuit elements arranged in parallel, the total impedance ΣZ_P is obtained by summing the reciprocals of individual impedances Z_{P1} and Z_{P2}:

$$\Sigma \frac{1}{Z_P} = \frac{1}{Z_{P1}} + \frac{1}{Z_{P2}} \quad \ldots\ldots\ldots (4)$$

The reciprocal of impedance is known as the admittance, Y.
Thus:

$$\Sigma Y_P = Y_{P1} + Y_{P2} \quad \ldots\ldots\ldots (5)$$

Defining admittance in terms of the j operator

$$Y = \frac{R}{R^2 + X^2} + j \frac{X}{R^2 + X^2} \quad \ldots\ldots\ldots (6)$$

$$\text{or} \quad Y = G + j B \quad \ldots\ldots\ldots (7)$$

If parallel circuit elements are written in the format of equation (7), these also may be added directly, and finally reconverted to an equivalent series impedance by use of equation (8):

$$Z = \frac{G}{G^2 + B^2} + j \frac{B}{G^2 + B^2} \quad \ldots\ldots\ldots (8)$$

Complex analysis consists in proceeding step by step with the addition of individual series and parallel elements in the manner described above until the entire circuit has been accounted for.

APPENDIX III to P.T.P. 829

The damping of oscillations

The behaviour of any oscillating system, electrical or mechanical, when subjected to a sudden constraint is conveniently described in terms of a "damping ratio". This is most usually calculated as the ratio of the observed damping to the values yielding critical damping. In an electrical circuit, the degree of damping is dependent upon the relative values of R (series resistance), L (series inductance) and C (series capacitance). Critical damping occurs when $R/2L = \sqrt{1/LC}$, and the damping ratio d is given by $\frac{R}{2}\sqrt{\frac{C}{L}}$ ..... (1)

In a mechanical system, where R, C, and L are not known with the same precision, it is sometimes preferable to calculate a logarithmic decrement ratio from the amplitude x_o and x of two successive half waves:

$$\text{Log decrement ratio} = \log_e \frac{x}{x_o} \qquad \ldots\ldots\ldots (2)$$

The relationship of the two measures of damping may be explained as follows. It is known (25) that

$$\log_e \frac{x}{x_o} = \frac{R}{2L} \cdot \frac{\pi}{f'} \qquad \ldots\ldots\ldots (3)$$

Thus

$$\frac{x}{x_o} = e^{-\frac{R\pi}{2Lf'}} \qquad \ldots\ldots\ldots (4)$$

Now the frequency of oscillation f is an undamped system is

$$f = \sqrt{\frac{1}{LC}} \qquad \ldots\ldots\ldots (5)$$

In a lightly damped system, the frequency f' is proportionately less

$$f' = \sqrt{f^2 - \left(\frac{R}{2L}\right)^2} \qquad \ldots\ldots\ldots (6)$$

$$= \sqrt{\frac{1}{LC} - \frac{R^2}{4L^2}} \qquad \ldots\ldots\ldots (7)$$

$$= \sqrt{\frac{4L - R^2C}{4L^2C}} \qquad \ldots\ldots\ldots (8)$$

Thus $$\frac{x}{x_o} = e^{-\frac{R\pi}{2L}} \sqrt{\frac{4L^2C}{4L-R^2C}} \qquad \ldots\ldots\ldots (9)$$

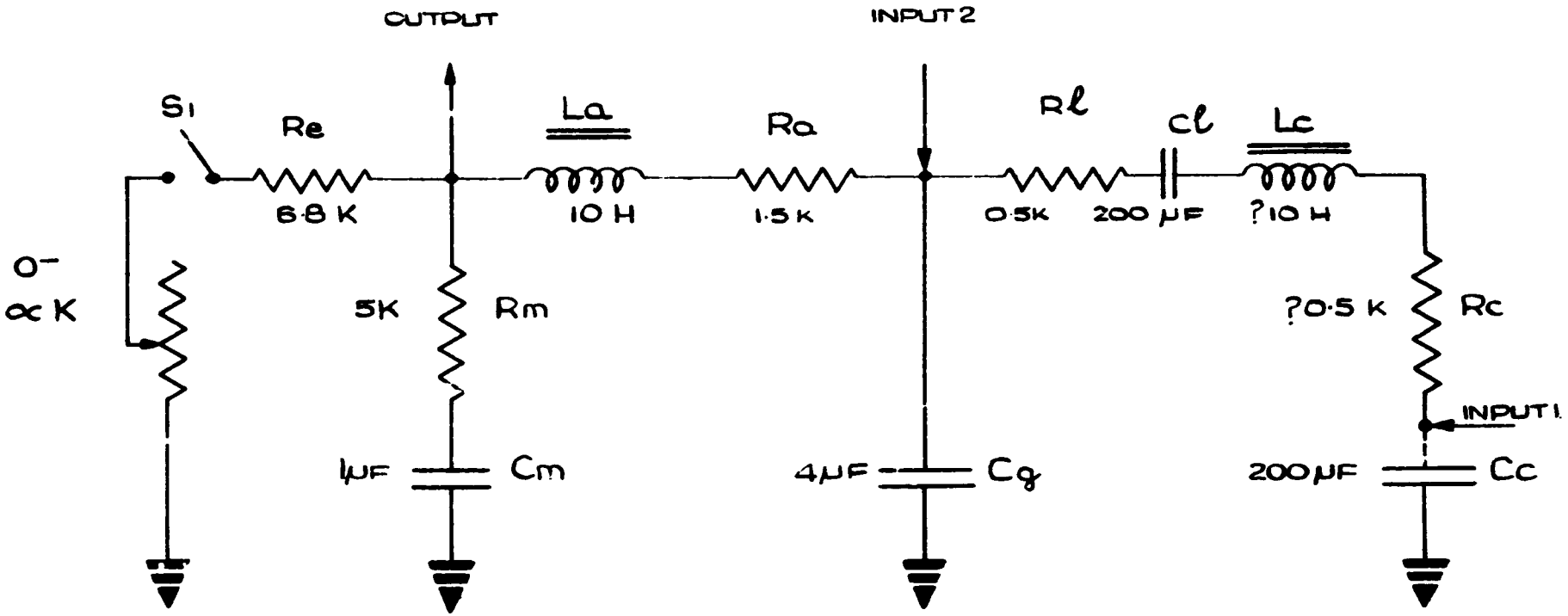
Now $$\frac{d^2}{1-d^2} = \frac{R^2C}{4L - R^2C} \qquad \ldots\ldots\ldots (10)$$

or $$\frac{R^2}{4L^2}\left(\frac{4L^2C}{4L - R^2C}\right) \qquad \ldots\ldots\ldots (11)$$

substituting in (1)

$$\frac{x}{x_o} = e^{-\frac{\pi}{\sqrt{1-d^2/d^2}}} \qquad \ldots\ldots\ldots (12)$$

FIG.1



ELECTRICAL ANALOGUE OF RESPIRATORY SYSTEM.

SCALE.

R : 1 cm H_2O / l / SEC = 1K

C : 0·001 l / cm H_2O = 1μF

L : 0·001 cm H_2O / l / SEC² = 1H

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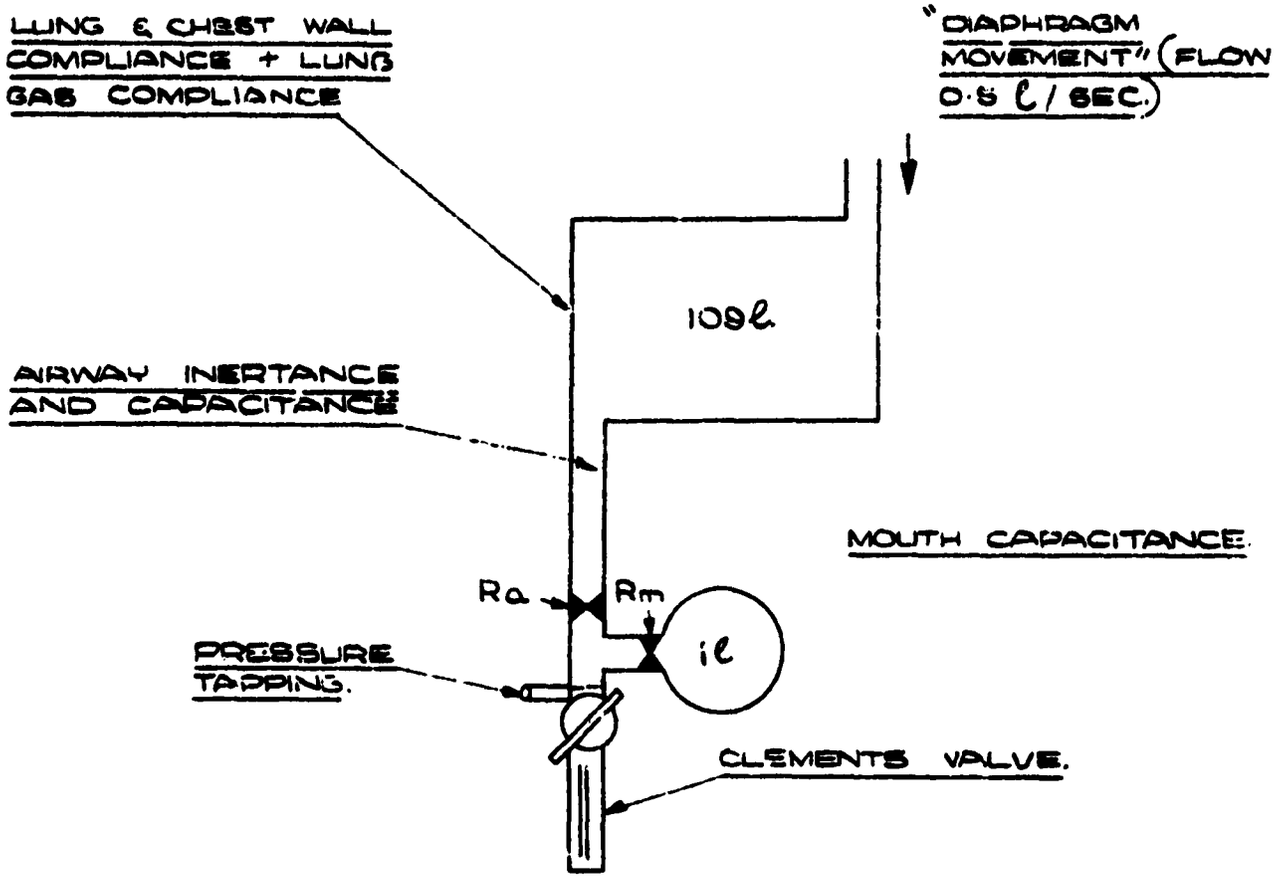
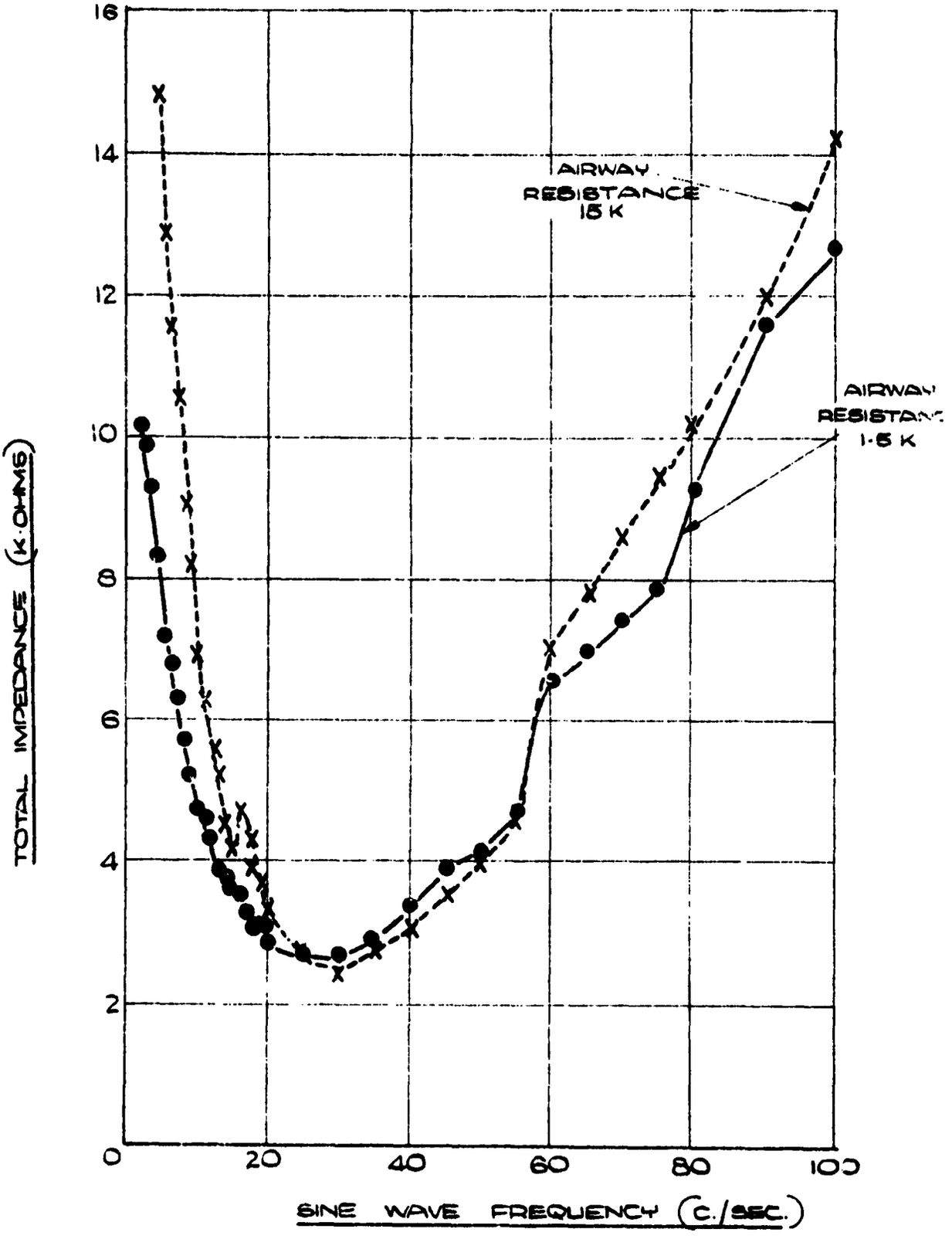


DIAGRAM OF MECHANICAL ANALOGUE OF RESPIRATORY SYSTEM

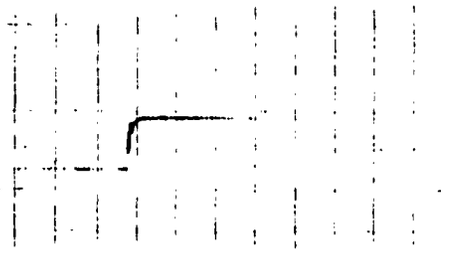
FIG. 2.

NOT TO SCALE

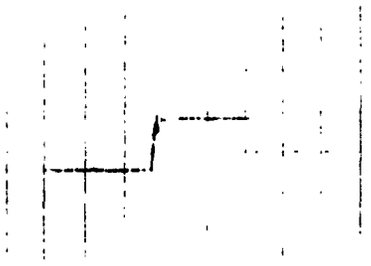


IMPEDANCE OF ELECTRICAL ANALOGUE OF RESPIRATORY SYSTEM. SINE WAVE APPLIED AT INPUT I OF FIG 1.

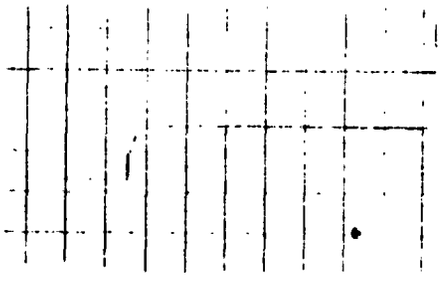
FIG.3



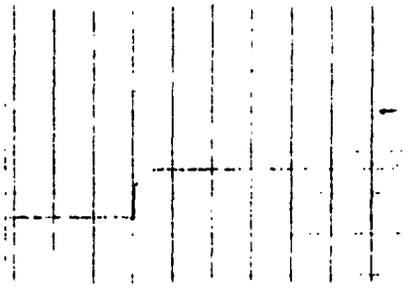
(a) NORMAL RESISTANCE (ANALOGUE)



(b) LOW MOUTH RESISTANCE.



(c) INCREASED AIRWAY RESISTANCE

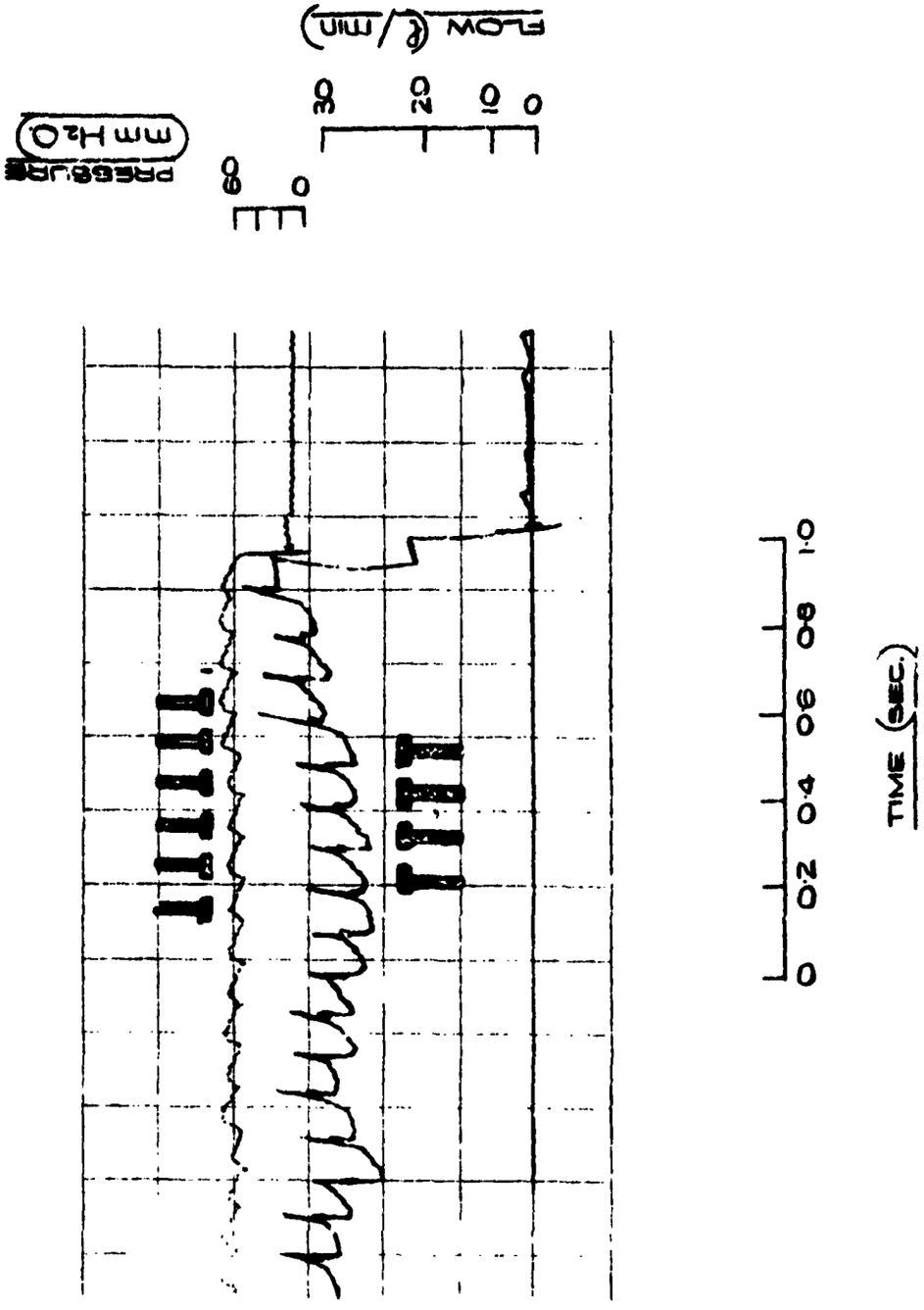


(d) NORMAL RESISTANCE, INCREASED MOUTH COMPLIANCE.

RESPONSE OF ELECTRICAL ANALOGUE TO SUDDEN OPENING OF EARTHING CONNECTION OF EXTERNAL RESISTANCE Re, CONSTANT POTENTIAL APPLIED TO LUNG GAS Cg PAPER SPEED 6 cm/sec.

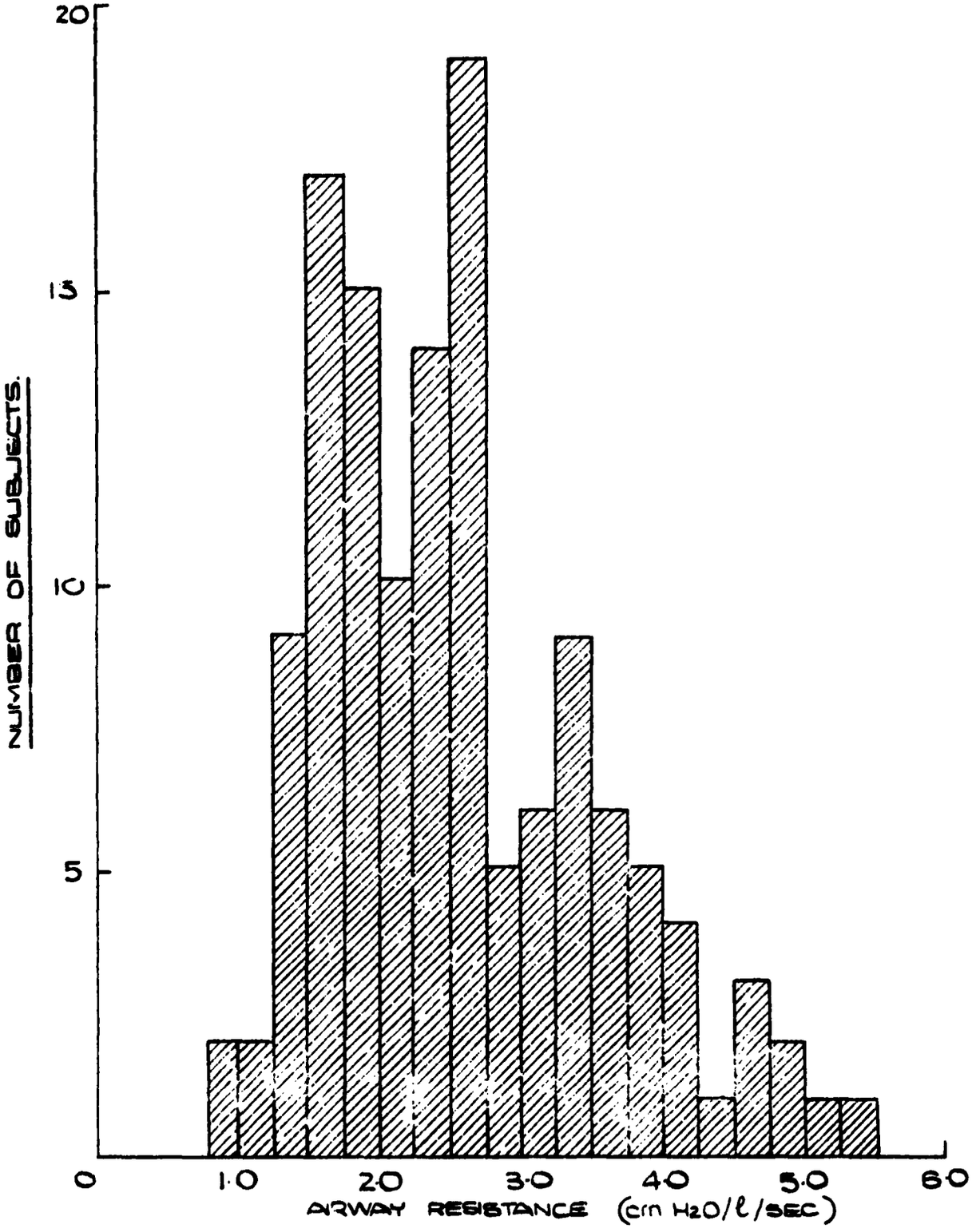
(a) NORMAL ANALOGUE SETTINGS
(b) MOUTH RESISTANCE 0·5 K
(c) AIRWAY RESISTANCE 15 K
(d) AIRWAY RESISTANCE 15 K, MOUTH COMPLIANCE 2 μF

FIG. 4



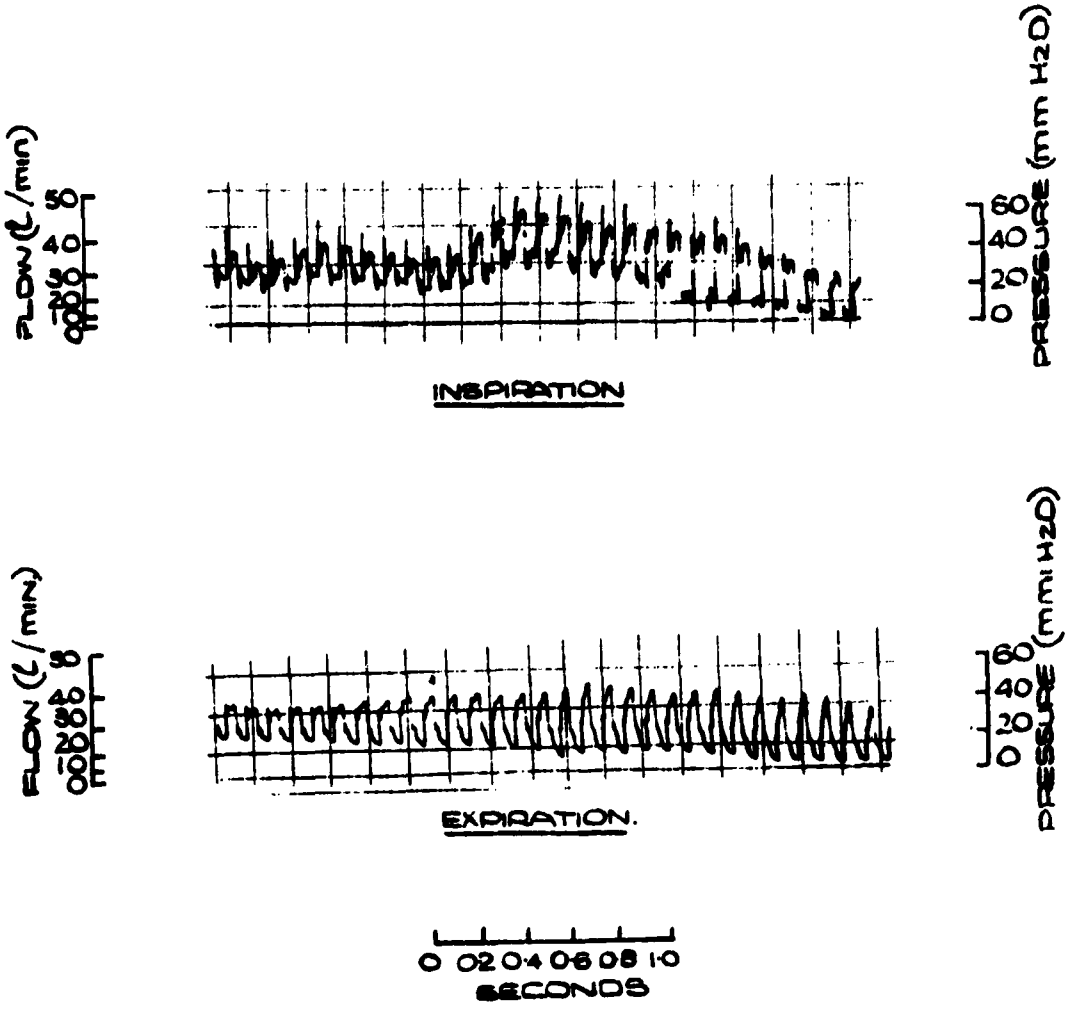
"SCANNED" RECORD OF "INTERRUPTION" AND "FLOW" PRESSURES FROM CLEMENTS INTERRUPTER VALVE. BARS INDICATE (a) INTERRUPTION AND FLOW PHASES OF CYCLE, AND (b) PORTION OF EACH PHASE IT IS INTENDED THAT THE SCANNING DEVICE SHALL SEE.

FIG.5



DISTRIBUTION OF AIRWAY RESISTANCE VALUES IN 132 SUBJECTS TESTED BY STANDARD CLEMENTS INTERRUPTER VALVE TECHNIQUE

FIG.6

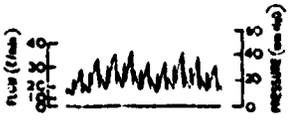


UNSCANNED RECORDS FROM CLEMENTS INTERRUPTER VALVE. TERMINAL HALF (a) OF MAXIMAL INSPIRATION, AND (b) MAXIMAL EXPIRATION.

FIG. 7

(a) NORMAL

(b) ADDED INERTANCE.



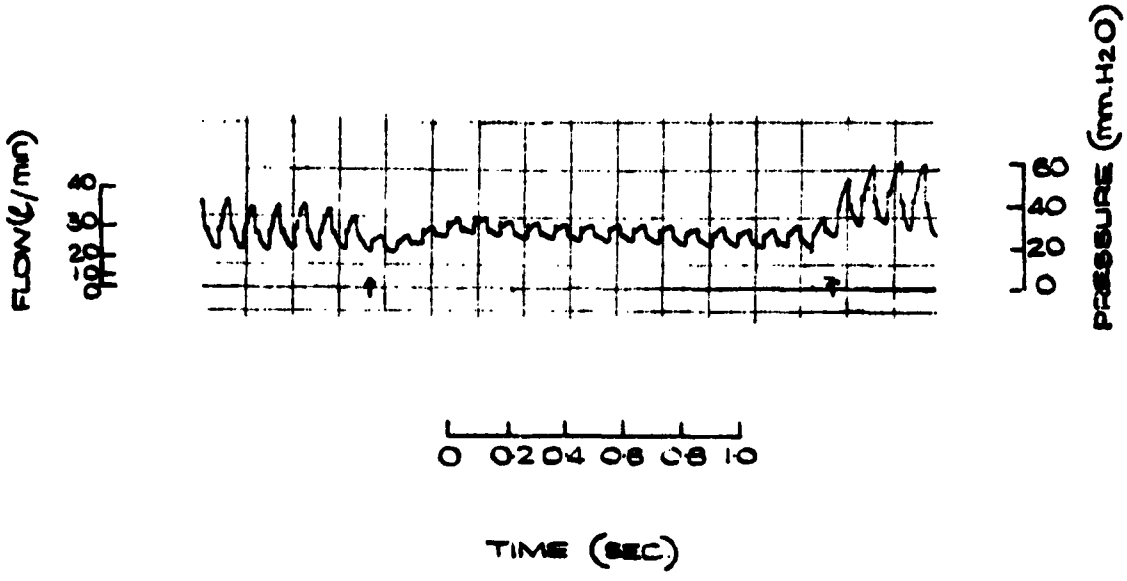
(c) LARYNGEAL CONSTRICTION WITH INERTANCE.

(d) CHRONIC BRONCHITIS.

(e) CHRONIC BRONCHITIS WITH INERTANCE.

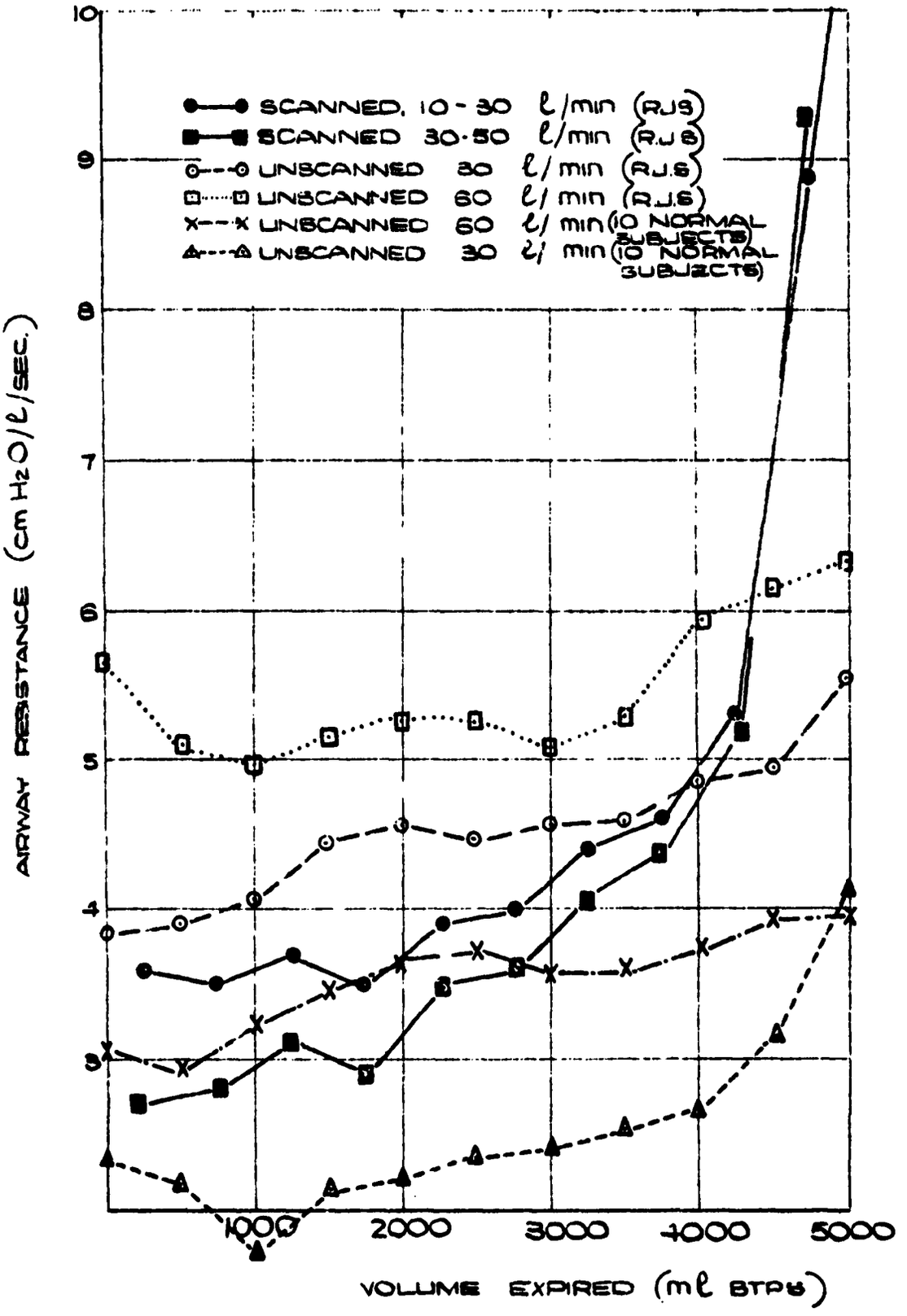
UNSCANNED RECORDS FROM CLEMENTS INTERRUPTER VALVE TO ILLUSTRATE EFFECT OF ADDING AN EXTERNAL INERTANCE (ADDED DEAD SPACE) IN NORMAL SUBJECTS AND IN SUBJECTS WITH INCREASED AIRWAY RESISTANCE (CONSTRICTION OF GLOTTIS OR CHRONIC BRONCHITIS)

FIG. 9



UNSCANNED RECORD FROM CLEMENTS INTERRUPTER VALVE (EXPIRATION). SUBJECT RELAXED CHEST WALL BETWEEN POINTS INDICATED BY ARROWS, MAINTAINING EXPIRATION BY ABDOMINAL MUSCLES.

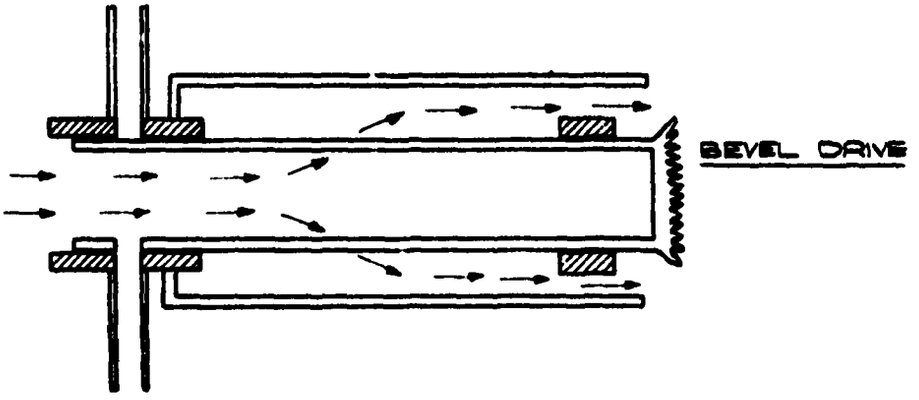
FIG. 9



AIRWAY RESISTANCE DURING STEADY MAXIMAL EXPIRATION.

FIG.10

"FLOW" PRESSURE TAPPING.



"PRESSURE" PRESSURE TAPPING.

DIAGRAM OF CLEMENTS INTERRUPTER VALVE (VALVE CLOSED). THE COURSE OF GAS FLOW WHEN THE VALVE IS OPEN IS INDICATED BY ARROWS.

FIG.11

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